Meth labs, in fact, have become known in law enforcement as the hazardous waste sites of the drug trade. "Almost every day, people are arrested who are willing to risk their lives and those of their children," says Thomas Abercombie, assistant laboratory director at the Bureau of Forensic Sciences in the California Department of Justice. "They usually don't have a clue what those chemicals can do to them."

In California, where meth manufacturing in the United States is largely centered, hospital admissions due to methamphetamine-related causes rose by 360% between 1986 and 1996. These patients included victims of fires, chemical spills, explosions, and the inhalation of toxic fumes. In December 1997, federal drug agents raided one meth lab in Los Angeles where the combustible and toxic chemicals were still cooking. Across the street was a day care center; nearby were two public schools.

Experts describe meth as the "poor man's cocaine"—a cheaper drug that gives people a longer high. "It's easy to manufacture," says Tom Cashman, a special agent with the United States Drug Enforcement Administration (DEA), who is the agency's leading expert on methamphetamine. "Amateurs can cook it up with a few chemicals in a makeshift lab. You don't need a lot of space or sophisticated equipment either. That's why it's being cooked in a variety of settings: apartments, cheap motels, mobile homes, and isolated farms and ranches."

It takes just \$4,000 in raw ingredients to make eight pounds of meth, which is worth \$50,000 on the street. Most of these ingredients are obtained from drug companies or the black market, or distilled from other substances, depending on the meth "cook"'s contacts and resourcefulness. While most of the chemicals needed are not

dangerous by themselves and can thus be easily obtained and manufactured, they create numerous environmental health hazards during the production process and after-



Cottage industry? Home labs (left) freqently use commonly available ingredients (right) in the production of illegal methamphetamine.

wards, when the chemicals are discarded.

Hydriodic acid and red phosphorus, the most dangerous chemicals used in meth production, can produce toxic phosphine gas and hydriodic acid vapors, while exposure to or inhalation of ether can cause respiratory damage, chemical burns, and even death. Red phosphorus poses additional problems because it's unstable and flammable, and can cause explosions and chemical fires if exposed to a flame or spark.

The fact that the meth-making process is getting easier has encouraged more people to get into the illegal activity. A recently unclassified DEA report reveals that phenyl-2-propanone (P2P) has been the primary precursor for meth manufacture. Since 1990, however, it has been increasingly replaced by the ephredine reduction method, a simpler procedure that involves fewer chemicals.

Rod Oswalt, a forensic scientist with the California Department of Justice, points out that the information for making meth is widely accessible. "The Internet contains thousands of recipes and discussions on how to make meth," he explains. "This, no doubt, has been a big factor in the rising popularity of meth."

The post-manufacturing phase poses the additional problem of what to do with the hazardous waste generated in making meth. One pound of finished meth normally produces 5–6 pounds of waste that includes corrosive sodium hydroxide solution, sealed cans containing residual freon and other hazardous fluids, and even pillow cases and bed sheets—used for filters—that hold large traces of red phosphorus and hydriodic acid, which can remain an environmental threat for years.

Cleaning up such hazardous waste sites is expensive, too. "It costs the taxpayers \$5,000–7,000 every time we clean up a

[meth lab] site," reveals Mike McCorson, an Arcadia, Californiabased hazardous waste coordinator for the Angeles National Forest.

Removing the containers, contaminated apparatus, and other typical waste is only a

part of the cleanup cost. Cleaning a building, for example, can involve removing carpet, washing the walls, and removing or cleaning the drywall and the wood stud framing. In one incident, the Los Angeles police found that meth manufacturers had used a garage adjoining a private residence. Due to the proximity of the lab, the contamination extended to the house. The final cost of cleanup was more than \$45,000.

Responsibility for cleanup costs is one of the biggest issues regarding methamphetamine, and varies from case to case, and state to state.

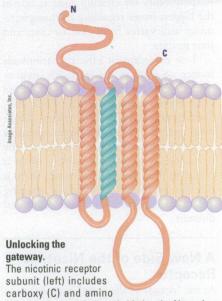
Law enforcement officials nationwide foresee no letup in the meth lab environmental problem. Says Rodney Pickel, an antinarcotics officer with the Rock Hill, South Carolina, police department, "Right now, meth is the drug of choice in mainly California, but that state usually sets the trend for the rest of the country when it comes to drug abuse." And where there is demand for drugs, supply follows, creating even more opportunities for environmental disasters.

A New Side of the Nicotinic Receptor

As the "decade of the brain" approaches an end, that wrinkly organ remains largely uncharted territory. But recent studies by two NIEHS researchers may lay important groundwork toward understanding the molecular mechanisms behind such neurological disorders as Alzheimer's disease, depression, epilepsy, and Parkinson's disease. Jerrel L. Yakel, a scientist in the Laboratory of Signal Transduction at the NIEHS, and Susan Jones, formerly of the NIEHS and now a postdoctoral fellow in the department of neurobiology at Duke University in Durham, North Carolina, have reported a study in the 1 November 1997 issue of the Journal of Physiology that reveals a previously unsuspected location and role for the brain's receptor for nicotine. By gaining a clearer understanding of the function of this receptor, it may be possible to devise more accurate methods for treating neurological disease.

The nicotinic receptor, one of two main categories of acetylcholine receptor, is a specialized ion channel that plays an important role in learning, memory, and survival- and stress-related responses. Ion channels function as "portholes" in the membranes of cells, opening or closing in response to chemical signals from outside, and therefore regulating the flow of electrical current through the cells. Says Yakel, "Our data provide a new mechanism to be considered about how the function of these ion channels, gated by the endogenous neurotransmitter acetylcholine or an exogenous activator [such as] nicotine, can act to regulate neuronal activity in a region of the brain—the hippocampus—that is believed to be a very important center for learning and memory."

Yakel's earlier work includes research on the role of the serotonin receptor in the brain. The Yakel-Jones study sought to



(N) termini; nicotinic ligands bind to the N terminus. The complete channel is thought to comprise five subunits clustered around a central ion "passageway" (right) that opens and closes like a camera shutter.

build upon that work by determining whether the nicotinic receptor would exhibit characteristics similar to those of the serotonin receptor. The scientists made an important discovery: nicotinic receptors, which had previously been thought to exist only on the presynaptic terminals of excitatory cells in the hippocampus, were also found on interneuronal (probably inhibitory) cells. The nicotinic receptor was thus found to act in a way previously unsuspected, possibly suggesting a novel mechanism by which acetylcholine may regulate neuronal activity in the hippocampus. This may further indicate that nicotinic ligands—the substances that bind to the nicotinic receptor—could be used as preventive measures against diseases associated with functions regulated by the receptor.

Neurological disease is a mysterious realm, with scientists' understanding limited to a few tantalizing bits of knowledge. It is known, for instance, that a mutation in one of the subunits of the nicotinic receptor is associated with the rare autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE), although whether this particular mutation actually causes this form of epilepsy has not been conclusively shown. It is also known that Alzheimer's patients have fewer nicotinic receptors in their cerebral cortex, the portion of the brain that is ravaged by the irreversible disease. But the relationships between the various components that make up the electrical circuit that powers the brain are largely undefined. Scientists such as Yakel and Jones are beginning to look at more specialized groups of cells in order to tease apart these relationships.

Theoretically, Yakel says, the ligands that bind with the nicotinic receptor could be used to treat diseases such as Alzheimer's disease. Nicotine has been observed to improve memory, including that of some Alzheimer's

patients. Little is known, though, about how nicotine actually works in the brain.

Some Alzheimer's patients are currently treated with a drug called

tacrine that works by blocking the enzyme acetylcholinesterase, which breaks down acetylcholine, therefore prolonging the period of time that neurons are exposed to the neurotransmitter. While the cellular activity of the drug appears clear, it is uncertain exactly why or how the drug works, and to what degree the nicotinic receptor is involved. Yakel says that further work with the nicotinic receptor will focus on ascertaining whether it has a direct link with Alzheimer's disease.

EDF Launches "3,000 by 2000" Project

Last year, the Environmental Defense Fund (EDF) released a study called *Toxic Ignorance* that found that the U.S. public does not have access to data on the basic health effects of 71% of high production volume chemicals. High production volume chemicals are defined by the EPA as those that are imported into or produced in the United States annually in quantities greater than 1 million pounds.

According to the Toxic Substances Control Act (TSCA) of 1976, the responsibility for testing chemicals for effects on health and the environment lies with the manufac-

turers of the chemicals. However, the EDF study asserts, up to this point, few efforts have been made by the chemical manufacturers to comply, and the government has not been effective in enforcing the mandates of the act. The EDF study states that this is due to the "self-defeating" language and structure of the TSCA, which cause it to be vulnerable to legal

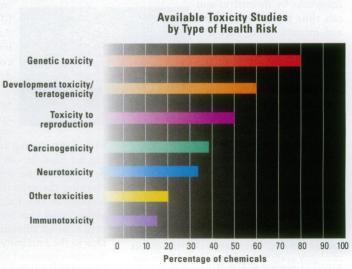
attacks by chemical manufacturers, thereby foiling the EPA's efforts to require chemical testing.

The EDF has begun a campaign to contact the chemical manufacturers directly, and challenge them to take responsibility and commit to testing chemicals. The goal of the campaign, called "3,000 by 2000," is to have health effects data available to the public for the top 3,000 high production volume chemicals by the year 2000.

The EDF sent letters to the top 100 chemical producers and challenged them to commit to completing a preliminary health screening on all chemicals that are produced in excess of 10 million pounds per year by 1 March 1999, and to screen all chemicals produced in excess of 1 million pounds per year by 1 January 2000. The EDF requested that the chemical manufacturers perform tests based on the human health hazard elements of the Screening Information Data Set (SIDS), developed in 1990 by the international Organisation for Economic Co-operation and Development (OECD) and the chemical manufacturing industry. SIDS is now considered the international standard for chemical testing. The six categories of potential adverse human health impacts are acute toxicity, repeated dose toxicity, in vitro genetic toxicity, in vivo genetic toxicity, developmental toxicity (including teratogenicity), and reproductive toxicity.

Eleven companies agreed to comply with the EDF's request, while six refused. Many others cited their current efforts to test chemicals. The EDF placed an advertisement in the 3 December 1997 issue of *USA Today* listing the companies that committed to the project, as well as those that refused.

By widely publicizing the campaign, the EDF has thrust the issue of chemical testing into the international spotlight,



Source: Toxic Ignorance. New York:Environmental Defense Fund, 1997